Remarks

In view of the above amendments and the following remarks, reconsideration of the outstanding office action is respectfully requested.

The objection to the title is respectfully traversed in view of the above amendment to the title.

The objection to claims 109-122 and 170-182 for being dependent on non-elected claim 104 is respectfully traversed in view of the above claim amendments.

The rejection of claims 109-113 and 115-119 under 35 U.S.C. § 101 as being directed to non-statutory subject matter is respectfully traversed in view of the above amendment to claim 109, which now recites an "isolated" detector.

The rejection of claims 109-113, 115-121, 170-175, and 177-181 under 35 U.S.C. § 112 (2nd para.) for indefiniteness is respectfully traversed in view of the above claim amendments.

The rejection of claims 109-122 and 170-182 under 35 U.S.C. § 112 (1st para.) for lack of enablement is respectfully traversed.

As set forth in the accompanying Declaration of Martin Frederick Pera Under 37 C.F.R. § 1.132 ("Pera Declaration"), the hybridoma which produces a GCTM-5 antibody has been deposited, under the terms of the Budapest Treaty, with the European Collection of Animal Cell Cultures (ECACC) Health Protection Agency, Porton Down, Salisbury, Wiltshire, SP4OJG, United Kingdom, under Accession No. 03101603. Pera Declaration, ¶ 4. This deposit was made on October 16, 2003. *Id.* All restrictions imposed by the depositor on the availability to the public of the deposited materials will be irrevocably removed as of the issue date of the above-identified patent application as a patent, pursuant to the conditions of 37 C.F.R. § 1.808. *Id.*

Therefore, this rejection is improper and should be withdrawn.

The rejection of claims 109-113, 115-121, 170-175, and 177-181 under 35 U.S.C. § 112 (1st para.) for lack of lack of written descriptive support is respectfully traversed.

The U.S. Patent and Trademark Office ("PTO") has asserted that detectors other than GCTM-5 antibody cannot be obtained without undue experimentation. However, contrary to the assertions of the PTO it is submitted that a person of ordinary skill in the art would be capable of identifying other detectors without undue experimentation in accordance with the

present claims. Specifically, the claims have been amended to refer to a detector which is capable of identifying a cell marker and where the cell marker is capable of being identified by a GCTM-5 antibody that is produced by hybridoma ECACC 03101603, or an active fragment thereof. In other words, the detector is characterized by sharing binding activity with the GCTM-5 antibody that is now fully characterized by reference to the hybridoma.

Therefore, the rejection of claims 109-113, 115-121, 170-175, and 177-181 under 35 U.S.C. § 112 (1st para.) for lack of written descriptive support is improper and should be withdrawn.

The rejection of claims 109-113 and 115-121 under 35 U.S.C. § 102(a) as anticipated by Schopperle et al., "Human Embryonal Carcinoma Tumor Antigen, Gp200/GCTM-2, is Podocalyxin," *Biochemical and Biophysical Research Communications* 300:285-290 (2003) ("Schopperle"), as evidenced by Bost et al., "Antibodies Against a Peptide Sequence Within the HIV Envelope Protein Crossreacts with Human Interleukin-2," *Immunological Investigations* 17:577-586 (1988) ("Bost") is respectfully traversed.

Schopperle reports that a peanut agglutinin-binding tumor antigen, gp200, a surface membrane glycoprotein expressed on human embryonal carcinoma, a malignant stem cell of testicular tumors, is similar to another embryonal carcinoma antigen, GCTM-2. GCTM-2 is a cell differentiation marker that is also detected in blood of testis cancer patients.

It is the position of the PTO in the outstanding office action that while Schopperle is silent with respect to a detector that binds to a stem cell marker characterized by binding to a GCTM-5 antibody, the antibodies recognized antigens derived from membrane preparation from testicular carcinomas. Thus, the PTO concludes, "similarity between both markers would allow for cross-reactivity of the antibodies." Office Action, pg. 7. In making this rejection, the PTO relies on Bost for the assertion that antibodies cross-react. Bost describes polyclonal antibodies that cross-react with IL-2 and HIV envelope protein. The binding of each protein in Bost is due to the presence of a homologous sequence in each protein in which 4-6 residues were identical.

However, applicants respectfully submit that Bost discusses the cross-reactivity of *polyclonal* antibodies (*see* Bost, pp. 578-9), whereas the antibodies disclosed in Schopperle are *monoclonal*. As a person of ordinary skill in the art would know, *polyclonal* antibodies, by

virtue of the manner in which they are generated, bind to multiple epitopes of the protein to which they have been generated, whereas *monoclonal* antibodies are directed to a single epitope.

The PTO has provided no other basis for its assertion that the antibodies disclosed in Schopperle bind the same surface membrane protein as the GCTM-5 antibody produced by hybridoma ECACC 03101603, as recited in the present claims. Neither can any such assertion be substantiated. There is no suggestion that the antibodies disclosed by Schopperle are directed to the same epitope as the GCTM-5 antibody. Even if the GCTM-5 antibody bound to the same cell type as the antibodies disclosed in Schopperle, the PTO has provided no evidence that the GCTM-5 monoclonal antibody of the present invention binds to a common epitope.

Since Schopperle does not teach or suggest each and every limitation of claims 109-113 and 115-121, the anticipation rejection based on this reference is improper and should be withdrawn.

The rejection of claims 109-113 and 115-121 under 35 U.S.C. § 102(b) as anticipated by Pera et al., "Analysis of Cell-Differentiation Lineage in Human Teratomas Using New Monoclonal Antibodies to Cytostructural Antigens of Embryonal Carcinoma Cells," *Differentiation* 39:139-149 (1988) ("Pera I"), as evidenced by Bost, is respectfully traversed.

Pera I produced monoclonal antibodies to cytostructurally associated antigens of human embryonal carcinoma cells. Monoclonal antibody *GCTM-1* stained the nuclei of all human cells tested and served as a positive control. This antibody immunoprecipitated proteins of 85 and 66 k Da from human embryonal carcinoma cells. *GCTM-2* recognized an epitope on a 200-k Da extracellular protein present on the surface of embryonal carcinoma cells. Antibody *GCTM-3* bound to a 57-k Da cytoskeletal protein expressed in human embryonal carcinoma. Antibody *GCTM-4* recognized a determinant present on a 69-k Da polypeptide, associated with a component of the lysosomal compartment, which was expressed in embryonal carcinoma cells, but no other cell type tested.

The PTO asserts that while Pera I is silent with respect to the antibodies binding to a stem cell marker characterized by binding to a GCTM-5 antibody, the antibodies disclosed in Pera I recognized antigens derived from membrane preparation from a testicular carcinoma with similar apparent molecular weight. According to the PTO, similarity between markers

would allow for cross-reactivity of the antibodies. Again, the PTO's position here is based on Bost's teaching of cross-reacting antibodies.

However, as noted above, Bost discusses the cross-reactivity of *polyclonal* antibodies. In contrast, the antibodies disclosed in Pera I are *monoclonal*. As a person of ordinary skill in the art would know, *polyclonal* antibodies, by virtue of the manner in which they are generated, bind to multiple epitopes of the protein to which they have been generated, whereas *monoclonal* antibodies are directed to a single epitope. Thus, the PTO's position of cross-reactivity is erroneous. Since the PTO has provided no other evidence that the antibodies disclosed in Pera I bind the same surface membrane protein as the GCTM-5 antibody produced by hybridoma ECACC 03101603, as recited in claims 109-113 and 115-121, the anticipation rejection based on this reference cannot be maintained and should be withdrawn.

The rejection of claims 109-113 and 115-121 under 35 U.S.C. § 102(e) as anticipated by PCT Publication No. WO 03/040355 to Pera et al. ("Pera II") or PCT Publication No. WO 01/98463 to Pera et al. ("Pera III"), as evidenced by Bost, is respectfully traversed.

Pera II and Pera III disclose various monoclonal antibodies.

The PTO has taken the position that while Pera II and Pera III are silent with respect to a detector that binds to a stem cell marker characterized by binding to a GCTM-5 antibody, the antibodies recognized surface membrane proteins expressed on human embryonic stem cells with similar apparent molecular weight and, therefore, similarity between the markers would allow for cross-reactivity of the antibodies, as described in Bost.

However, as noted *supra*, Bost discusses the cross-reactivity of *polyclonal* antibodies. In contrast, the antibodies disclosed in Pera II and Pera III are *monoclonal*. Thus, as noted above, the PTO's position of cross-reactivity cannot form a proper basis for this rejection. Since the PTO has provided no other evidence that the antibodies disclosed in Pera II and Pera III bind the same surface membrane protein as the GCTM-5 antibody produced by hybridoma ECACC 03101603, as recited in claims 109-113 and 115-121, the anticipation rejection based on these references cannot be maintained and should be withdrawn.

The rejection of claims 170-175 and 177-181 under 35 U.S.C. § 103(a) for obviousness over Schopperle, Pera I, Pera II, and Pera III, each in view of U.S. Patent No. 4,281,061 to Zuk et al. ("Zuk") is respectfully traversed.

The teachings of Schopperle, Pera I, Pera II, and Pera III are noted above.

Zuk is cited for teaching that reagents or pharmaceutical compositions can be provided as kits as a matter of convenience, optimization, and economy of the user.

According to the PTO, it would have been obvious for a person of ordinary skill in the art at the time of the invention to apply the teachings of Zuk to those of Schopperle, Pera I, Pera II, or Pera III to obtain a kit comprising a detector of a cell type which identifies on the cell type a cell marker, characterized by binding to a GCTM-5 antibody, as recited in the present claims.

However, since Zuk does not overcome the above-noted deficiencies of Schopperle, Pera I, Pera II, and Pera III, this rejection is improper and should be withdrawn.

In view of the foregoing, it is submitted that this case is in condition for allowance and such allowance is earnestly solicited.

Respectfully submitted,

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